In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: February 26, 2025

MITCHELL GODFREY,	*	PUBLISHED
Petitioner,	*	No. 17-1419V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal Decision; Pneumococcal Conjugate ("Prevnar 13") Vaccine; Guillain-Barré Syndrome ("GBS").
Respondent.	*	

Isaiah Kalinowski, Bosson Legal Group, Fairfax, VA, for Petitioner.
Jennifer Leigh Reynaud, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

On October 3, 2017, Mitchell Godfrey ("Petitioner"), filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018),[2] alleging that as a result of a pneumococcal conjugate vaccine ("Prevnar 13") administered on August 10, 2016, he developed Guillain-Barré Syndrome ("GBS"). Petition at ¶¶ 3-8 (ECF No. 1). Respondent argued against compensation, stating "compensation is not appropriate under the terms of the Vaccine Act." Respondent's Report ("Resp. Rept.") at 9 (ECF No. 22).

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018) ("Vaccine Act" or "the Act"). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

After carefully analyzing and weighing the evidence presented in accordance with the applicable legal standards,[3] the undersigned finds Petitioner failed to provide preponderant evidence that his Prevnar 13 vaccine caused him to develop GBS. Thus, Petitioner has failed to satisfy his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, the petition must be dismissed.

I. ISSUES TO BE DECIDED

The parties do not dispute Petitioner's diagnosis is GBS.[4] Petitioner's Motion for Findings of Fact and Conclusions of Law Regarding Entitlement to Compensation ("Pet. Mot."), filed Apr. 13, 2024, at 27 (ECF No. 98); Resp. Brief Opposing Entitlement ("Resp. Br."), filed June 21, 2024, at 17 (ECF No. 102) ("[R]espondent does not dispute [P]etitioner's diagnosis of GBS."); Pet. Reply Memorandum in Support of Pet. Mot. ("Pet. Reply Memo."), filed July 29, 2024, at 16 (ECF No. 104). Additionally, "the parties do not seem to meaningfully disagree as to the onset of [P]etitioner's GBS." Resp. Br. at 17.

At issue is whether Petitioner has provided preponderant evidence of all three Althen prongs. Pet. Mot. at 11-39; Resp. Br. at 17-30.

[3] While the undersigned has reviewed all of the information filed in this case, only those filings and records that are most relevant will be discussed. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision."); see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

[4] Although the parties and experts agree Petitioner's diagnosis is GBS, there is a disagreement as to the specific variant of GBS Petitioner developed. The undersigned does not discuss these arguments, or make any specific findings as to Petitioner's variant of GBS, because it is not pertinent to the undersigned's Decision. See Resp. Exhibit ("Ex.") A, Tab 1 at 5 ("Classification of [GBS] as either acute inflammatory demyelinating polyneuropathy [("AIDP")] or acute motor axonal neuropathy [("AMAN")] is not required for diagnosis of [GBS] . . . .") (Hugh J. Willison et al., Guillain-Barré Syndrome, 388 Lancet 717 (2016)); Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010) ("identifying [Petitioner's] injury is a prerequisite" to the Althen analysis but clarifying "[P]etitioner [is] not required to categorize his injury where the two possible diagnoses [are] 'variants of the same disorder'" (quoting Kelley v. Sec'y of Health & Hum. Servs., 68 Fed. Cl. 84, 100-01 (2005))).

II. BACKGROUND

A. Procedural History

Petitioner filed his petition on October 3, 2017 followed by medical records and affidavits in October and November 2017.[5] Petition; Pet. Exs. 1-9. On September 10, 2018, Respondent filed his Rule 4(c) report, arguing against compensation. Resp. Rept. at 1.

From April 2019 to February 2023, Petitioner filed expert reports from Dr. Zurab Nadareishvili and Dr. Lawrence Steinman and Respondent filed expert reports from Dr. Vinay Chaudhry and Dr. J. Lindsay Whitton.[6] Pet. Exs. 99, 101, 112, 142; Resp. Exs. A, C, E-F.

Thereafter, the parties agreed to resolve entitlement through a ruling on the record. Joint Status Rept., filed Feb. 9, 2024 (ECF No. 96). Petitioner filed his motion for a ruling on the record on April 13, 2024. Pet. Mot. Respondent filed his responsive brief on June 21, 2024 and Petitioner filed a reply on July 29, 2024. Resp. Br.; Pet. Reply Memo.

This matter is now ripe for adjudication.[7]

B. Medical Terminology

GBS is a "rapidly progressive ascending motor neuron paralysis" that typically "begins with paresthesias of the feet, followed by flaccid paralysis of the entire lower limbs, ascending to the trunk, upper limbs, and face; other characteristics include slight fever, bulbar palsy, absent or lessened tendon reflexes, and increased protein in the cerebrospinal fluid [("CSF")] without a corresponding increase in cells." Guillain-Barré Syndrome, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=110689 (last visited Feb. 7, 2025). Although the etiology of GBS is unknown, it is "frequently seen after an enteric or respiratory infection. An autoimmune mechanism following viral infection has been postulated." Id.; see also Resp. Ex. A, Tab 1 at 1-2 ("[GBS] is usually preceded by infection or other immune stimulation that induces an aberrant autoimmune response targeting peripheral nerves and their spinal roots."); Pet. Ex. 61 at 2 ("Although the underlying etiology and pathophysiology of GBS are not completely understood, it is believed that immune stimulation plays a central role in its pathogenesis.").[8]

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[5] Medical records were filed throughout litigation.

[6] Prior to Petitioner's first expert report in April 2019, Petitioner's counsel filed a large amount of literature, most of which was not cited or discussed by either expert. See Pet. Exs. 12-98.

[7] This case was reassigned to the undersigned on August 29, 2024. Notice of Reassignment dated Aug. 29, 2024 (ECF No. 110).

[8] James J. Sejvar et al., Guillain-Barré Syndrome and Fisher Syndrome: Case Definitions and Guidelines for Collection, Analysis, and Presentation of Immunization Safety Data, 29 Vaccine 599 (2011).

"[A]pproximately two-thirds of persons with GBS report an antecedent infectious illness, most commonly a diarrhoeal or respiratory illness, in the days or weeks preceding neurologic signs." Pet. Ex. 61 at 3; see also Pet. Ex. 35 at 2 (noting "about two-thirds of [GBS] patients have had an infection within the previous [six] weeks," including gastroenteritis);[9] Pet. Ex. 38 at 2-4, 4 fig.1 (noting "[t]wo-thirds of GBS patients have histories of antecedent infectious illness" and finding 29% (51/176) of their patients had a gastrointestinal ("GI") illness prior to onset of GBS and 27% (47/176) reported diarrhea);[10] Pet. Ex. 89 at 2 ("Some form of infection was found to precede nearly 90% of GBS cases, with symptoms of upper respiratory infection in 60% and of GI illness in 30%.").[11] "Often, the responsible organism is not identified." Pet. Ex. 35 at 2.

"Although several microorganisms have been associated with GBS development, *Campylobacter jejuni* [("*C. jejuni*")] is the most extensively studied pathogen as it is a common antecedent to GBS." Pet. Ex. 26 at 6-7;[12] see also Pet. Ex. 61 at 3; Pet. Ex. 38 at 1 ("*C. jejuni* is the most common antecedent infectious agent in GBS."); Pet. Ex. 52 at 9 ("*C. jejuni* infection is the predominant antecedent infection in GBS. It has been identified in 30%-50% of GBS patients . . . ").[13] *C. jejuni* is "a species that is a common cause of enteric campylobacteriosis in humans." Campylobacter Jejuni, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=62516 (last visited Feb. 7, 2025).[14]

Lastly, "[c]ases of [GBS] have also been reported shortly after vaccination." Resp. Ex. A, Tab 1 at 2; see also Pet. Ex. 26 at 6 (noting "[o]nset of GBS occurs days or weeks following an infection or immunization"); Pet. Ex. 52 at 8-9.

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[9] Richard A.C. Hughes & David R. Cornblath, Guillain-Barré Syndrome, 366 Lancet 1653 (2005).

[10] M. Koga et al., Antecedent Symptoms in Guillain-Barré syndrome: An Important Indicator for Clinical and Serological Subgroups, 103 Acta Neurologica Scandinavica 278 (2001).

[11] Nobuhiro Yuki, Ganglioside Mimicry and Peripheral Nerve Disease, 35 Muscle & Nerve 691 (2007).

[12] A.M. Ercolini & S.D. Miller, The Role of Infections in Autoimmune Disease, 155 Clinical & Experimental Immunology 1 (2008).

[13] Kishan Kumar Nyati & Roopanshi Nyati, Role of *Campylobacter jejuni* Infection in the Pathogenesis of Guillain-Barré Syndrome: An Update, 2013 BioMed Rsch. Int'l 1.

[14] For more information on *C. jejuni*, including clinical manifestations, diagnosis, and treatment, see Resp. Ex. A, Tab 6 (Ban M. Allos, Clinical Manifestations, Diagnosis, and Treatment of Campylobacter Infection, UpToDate, https://www.uptodate.com/contents/campylobacter-infection-clinical-manifestations-diagnosis-and-treatment (last updated Aug. 9, 2019)).

C. Summary of Relevant Medical Records

On August 10, 2016, at 71 years of age, Petitioner received a Prevnar 13 vaccination through the Veterans Association ("VA") at Fort Harrison Medical Center ("Fort Harrison"), the office of his primary care physician ("PCP") Dr. Jaye Swoboda, during an annual visit. Pet. Ex. 1 at 1; Pet. Ex. 3 at 157. Dr. Swoboda documented Petitioner felt well and Petitioner had no bowel or bladder changes. Pet. Ex. 3 at 157. Petitioner's prior medical history did not include neurological complaints. See Pet. Mot. at 2; Resp. Br. at 3-4.

Approximately 19 days later, on Monday, August 29, 2016, Petitioner returned to Fort Harrison as a walk-in, complaining of "feeling puffy at the bottom of the feet," which "fe[lt] like walking on gel, left more pronounced tha[n] right." Pet. Ex. 3 at 51. Petitioner reported his symptoms began four days prior (August 25).[15] Id. Petitioner requested an appointment with his PCP. Id. Registered nurse James Montana's physical examination was unremarkable; Petitioner had no nausea, vomiting, or bowel issues. Id. The "[i]dentified problem" was documented as "foot inflammation, unspecified." Id. at 152.

Petitioner saw his PCP at Fort Harrison the next day, August 30. Pet. Ex. 3 at 147. Petitioner reported diffuse myalgias without fever beginning Saturday (August 27). Id. at 148. Petitioner reported he "[felt] like he [was] walking on [pads] over metatarsal heads." Id. He was considering cancelling a planned trip to Salt Lake City. Id. On examination, Dr. Swoboda documented Petitioner's quadriceps were tender, Petitioner appeared tired and had trouble making eye contact, and that Petitioner's "[f]eet [were] normal by brief exam[ination] but very cold to touch." Id. Assessment was diffuse myalgias of an unknown etiology. Id. Dr. Swoboda wrote "[t]his could represent delayed reaction to [Prevnar 13]." Id. Bloodwork for creatine phosphokinase and erythrocyte sedimentation rate were ordered and normal. Id. at 147-48.

A few days later, on September 2, 2016, Petitioner returned to Fort Harrison "reporting tingling/numbness of feet and []9/10 pain of rest of body [for] [one] week" despite taking Tylenol and Aleve. Pet. Ex. 3 at 145. Petitioner was diagnosed with neuropathy and prescribed gabapentin 300 mg. Id.

The following day, September 3, Petitioner presented to the emergency department at Bozeman Deaconess Hospital. Pet. Ex. 6 at 4. Emergency physician Dr. Leslie Cohen documented Petitioner's clinical course: "Approximately one week ago the patient developed tingling on the bottoms of both of his feet. This was followed [two] days later by balance issues and mild leg weakness. . . . He then developed diffuse body aches and generalized malaise. Yesterday he developed severe mid and low back pain." Id. Petitioner underwent "a lumbar puncture [for] further evaluation of [GBS]." Id. Petitioner underwent a lumbar puncture but declined admission following the procedure and prior to receiving the results. Id. Petitioner was then advised to return for magnetic resonance imaging ("MRI") of his brain, thoracic spine, and lumbar spine. Id. Dr. Cohen's note upon Petitioner's return documented Petitioner's "back pain ha[d] significantly improved [with] minimal pain," but "his gait still fe[lt] off and . . . not normal." Id. at 4-5. Petitioner denied progression of weakness or change in sensation on the

[15] This would place symptom onset on Thursday, August 25, 2016, 15 days post-vaccination.

bottoms of his feet. Id. at 5. Dr. Cohen documented, "Patient reports diarrhea several weeks ago." Id. Petitioner denied diarrhea currently as well as fever, chills, chest pain, shortness of breath, abdominal pain, nausea, vomiting, inability to urinate, bowel dysfunction, dizziness, or syncope. Id. On physical examination, Dr. Cohen documented Petitioner had a "slightly broad-based gait" although "[h]e [was] able to ambulate without difficulty." Id. at 6-7. Petitioner also had "absent patellar reflexes bilaterally." Id. at 7. Sensations of lower extremities and buttocks and his lower extremity motor function were intact and he was "able to dorsiflex his great toe bilaterally." Id. at 6-7.

Petitioner's CSF showed elevated protein (84.7; reference range 15-60) and normal glucose (59; reference range 40-75), as well as red blood cells (reference range 0), normal white blood cells (2; reference range 0-5), and elevated lymphocytes (95%; reference range 40-80%). Pet. Ex. 6 at 7-8. Petitioner's brain MRI, with and without contrast, revealed "[s]cattered periventricular and subcortical white matter punctate foci of T2/FLAIR hyperintensity," which were "nonspecific findings" that could be "normal age-related changes, or very minimal chronic microvascular ischemic change (especially there is a history of diabetes, dyslipidemia, smoking, or hypertension). Much less likely considerations would include demyelinating phenomena and such as multiple sclerosis [("MS")], Lyme disease, vasculitis, or [acute disseminated encephalomyelitis ("ADEM")]." Id. at 8-9. Petitioner's thoracic spine MRI, with and without contrast, revealed a "[t]iny disc bulge at T5-6, resulting in minimal ventral cord compression," but "[n]o associated cord signal abnormalities or abnormal thoracic cord enhancement;" a "[s]mall disc osteophyte complex at C7-T1" with "no cord deformity;" and "[n]o significant neuroforaminal stenosis." Id. at 10-11. Lastly, impression of Petitioner's lumbar spine MRI, with and without contrast, was "[m]ild to moderate lumbar spondylosis" and "[n]o abnormal lumbar spine, lumbar cord[,] or spinal nerve root enhancement." Id. at 11-13.

Dr. Cohen opined the "MRIs were relatively unremarkable and [did] not account for [Petitioner's] symptoms." Pet. Ex. 6 at 14. She consulted with neurologist, Dr. Jozef Ottowicz, from Billings Clinic, who reviewed Petitioner's presentation, history, physical examination, CSF, and MRIs and determined Petitioner likely had GBS. Id. at 13-14. Dr. Ottowicz recommended nerve conduction studies ("NCS") and five days of IVIG. Id. Petitioner agreed and was admitted. Id.

On admission on September 3, Petitioner saw hospitalist Dr. Tory Katz. Pet. Ex. 6 at 20. Petitioner reported "that over [two] weeks ago he had a brief diarrheal illness that has completely resolved." Id. And approximately one week ago, he developed numbness in his feet and gait and balance issues. Id. Petitioner reported "[h]e is usually very active," hiking three times a week and playing 18 holes of golf at least twice a week, but he has not been able continue these activities. Id. Physical examination conducted by Dr. Katz documented absent deep tendon reflexes in his patellar, biceps, and Achilles tendon. Id. at 22. Dr. Katz opined that "[g]iven recent history of a diarrheal illness followed by development of the [reported] symptoms[,] [I] feel this is consistent with [GBS]." Id. at 23. Assessment was "mild ascending flaccid paralysis concerning for [GBS]." Id.

On September 6, 2016, Petitioner was seen by neurologist Dr. Joshua Knappenberger for a consultation. Pet. Ex. 6 at 24. Dr. Knappenberger documented Petitioner "developed a mild

GI illness with diarrhea for a couple of days" one month ago, without fever or abdominal pain. Id. He also documented Petitioner received a Prevnar 13 vaccination "about [three] weeks ago." Id. Petitioner first developed symptoms of numbness and paresthesias "one week ago on Saturday" while golfing. Id. Petitioner's symptoms began to ascend up to his calves bilaterally. Id. He was prescribed gabapentin but reported he took only one dose prior to presenting to the emergency department. Id. Since starting IVIG, Petitioner "felt like he ha[d] been gradually getting better, with improved balance." Id. On September 5, Petitioner developed some irritation in his right eye, and on September 6, he had right facial weakness. Id. Due to this facial weakness, Petitioner developed issues with speech, specifically trouble enunciating words. Id.

Dr. Knappenberger's physical examination of Petitioner's cranial nerves documented right facial droop and issues closing his right eye. Pet. Ex. 6 at 26. Sensory examination revealed "[d]ecreased vibration to the knees, decreased to temperature and pinprick, light touch to mid shin and mid forearm." Id. Petitioner's deep tendon reflexes were 2+ in his arms and absent in the ankles. Id. Petitioner also had "[a] little wide based, unsteady tandem" gait. Id. Dr. Knappenberger's assessment was GBS. Id. He based his opinion on Petitioner's paresthesias and unsteadiness on his feet, which was bilateral and subacute; Petitioner's back pain, "which is quite common in [GBS];" Petitioner's physical examination showing "hyperreflexi[a] in the legs with vibratory sense diminished out of proportion to his other modalities;" and albuminocytologic dissociation[16] in his CSF. Id. Petitioner was to continue IVIG, physical therapy,[17] occupational therapy, and speech therapy and was directed to obtain an out-patient electromyography ("EMG"). Id. With regard to the right facial weakness, Dr. Knappenberger assessed Petitioner with an "incomplete right seventh cranial nerve palsy associated with [GBS]." Id. at 27. He ordered an MRI to rule out stroke "given that unilateral facial nerve weakness is fairly uncommon in [GBS]," IVIG can increase coagulation, and Petitioner's speech problems. Id. Lastly, Dr. Knappenberger ordered a cervical spine MRI due to Petitioner's "preserved arm reflexes with sensory changes in the arms." Id.

Petitioner's second brain MRI on September 6 revealed "[u]nremarkable trigeminal nerves," "[n]o evidence of acute intracranial pathology," stable "[s]cattered nonspecific areas of high T2 and FLAIR signal in the white matter" that were "most consistent with chronic microangiopathy." Pet. Ex. 6 at 16. And his cervical spinal MRI showed "[m]ultifactorial

[16] Albuminocytologic dissociation refers to the "increase of protein with normal cell count in the spinal fluid" and is a classic characteristic seen in GBS. Albuminocytologic Dissociation, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=71273 (last visited Feb. 7, 2025); see Guillain-Barré Syndrome, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=110689 (last visited Feb. 7, 2025); Resp. Ex. A, Tab 1 at 5-6.

[17] Petitioner continued to receive physical therapy from September 2016 to January 2018 to improve his balance, strength, and conditioning. See, e.g., Pet. Ex. 3 at 132-33, 339-45; Pet. Ex. 7 at 1-50; Pet. Ex. 10 at 5-33; Pet. Ex. 11 at 19, 58, 61, 66, 82-86; Pet. Ex. 84 at 57, 61, 64, 73, 208, 455-64, 470-74.

spondylosis . . . most pronounced at C5-C6 and C6-C7 where there is mild to moderate spinal canal narrowing and mild neural foramen narrowing. No abnormal enhancement." Id.

Petitioner was discharged on September 7, 2016 following five days of IVIG. Pet. Ex. 6 at 15-16. His discharge diagnoses were GBS and incomplete peripheral seventh cranial nerve palsy due to GBS. Id. at 15.

Petitioner saw his PCP Dr. Swoboda on September 16, 2016. Pet. Ex. 3 at 138. Petitioner reported 2/10 pain and that he had "persistent weakness and ache in legs which [was] relieved by hydrocodone." Id. Dr. Swoboda noted Petitioner was walking with a cane, was "a bit wobbly," and could stand on one leg. Id. Dr. Swoboda "strongly suspect[ed] GB[S] due to [Prevnar 13]." Id. at 55. Dr. Swoboda "[r]eported possible [Prevnar 13] etiology to pharmacy."[18] Id. at 138. He also listed Prevnar as an allergy/adverse reaction and "deferred [influenza ("flu")] vaccine indefinitely." Id. at 56, 120.

On September 21, 2016, Petitioner followed up with Dr. Knappenberger. Pet. Ex. 2 at 24. Petitioner's face was "somewhat better." Id. Petitioner "continue[d] to have numbness and tingling in the feet, up to the balls of the feet, to the heel on the left," but "[n]ot above the ankles." Id. He also had a wide-based gait, headache, and "[a] little bit of [back] pain." Id. Neurologic examination revealed "[f]ace [was] symmetrical without apparent sensory loss or weakness;" an abnormal sensory examination that displayed decreased sensations; deep tendon reflexes of 1+ in the arms and absent in the legs; and wide-based gait (four inches) and effort with tandem gait. Id. at 26-27. Assessment was "[GBS] following vaccination." Id. at 27. Dr. Knappenberger noted Petitioner's cranial nerve palsy was "markedly improved" as was his distal sensory loss. Id. Petitioner's legs remained hyperreflexic with some distal sensory change. Id. Dr. Knappenberger ordered an EMG for the following day. Id.; Pet. Ex. 85 at 2.

Petitioner returned to Dr. Knappenberger on October 13, 2016 and reported continued back pain and tightness, headaches, and "little tingling" in hands and feet with numbness in feet spreading throughout the day. Pet. Ex. 2 at 46. Physical examination revealed decreased sensations, trace deep tendon reflexes in the biceps, absent deep tendon reflexes in the legs, normal stance with narrow base and symmetrical arm swing, and stable tandem gait. Id. at 48-49. Assessment remained "[GBS] following vaccination." Id. at 49. Dr. Knappenberger noted Petitioner "[c]ontinue[d] to improve with decreasing neuropathic symptoms with the exception

[18] Pharmacist Keaten Labrel responded and "strongly encourage[d] this adverse event be reported" to Vaccine Adverse Event Reporting System ("VAERS"). Pet. Ex. 3 at 137. Pharmacist Labrel noted "neither the [Centers for Disease Control and Prevention ("CDC")] or [World Health Organization ("WHO")] have reported the incidence of GBS secondary to [P]revnar-13 immunizations due to there not being enough evidence [to] support causal association." Id. Furthermore, "[t]here are very few post-marketing analysis reports that illustrate development of GBS or sixth cranial nerve palsy after [Prevnar 13] administration; however there are reports of development of GBS." Id. Thereafter, the pharmacy entered a VAERS report on Petitioner's behalf in December 2016. Id. at 43. The undersigned was unable to find this VAERS report in the record.

of his back pain," which Dr. Knappenberger believed was a "muscle spasm related to radicular irritation with his [GBS]." Id.

On December 1, 2016, Petitioner followed up with Dr. Knappenberger. Pet. Ex. 2 at 59. Petitioner did not have any new facial weakness. Id. He reported an issue with vision due to his gabapentin. Id. He also reported his hands felt "a little fuzzy" and "a bit numb on the tips," but there was no weakness or pain. Id. Petitioner continued to report numbness in his feet and back pain. Id. Assessment was "[p]arethesias. EMG with evidence of acute radiculoneuropathy, non-length dependent exam[ination]. . . . This seems to be monotonic [GBS], and his main issue now is residual back pain which may be from radicular irritation although mechanical back pain is certainly a possibility as well." Id. at 62. Dr. Knappenberger noted Petitioner's MRIs "showed no mass lesion" and "some degeneration." Id.

At Petitioner's next visit with Dr. Knappenberger on February 2, 2017, Petitioner reported continued numbness, described as "fuzzy," in the pads of his feet that did not improve since his last visit; continued "fuzzy" feeling in tips of fingers; ankles were getting stronger; back pain was "pretty well,"[19] with "a lot of back itching" and continued improvement with sharp pain; and no headaches, diplopia, facial numbness, or facial weakness. Pet. Ex. 2 at 101. Petitioner reported using a walking stick and that his balance "[was] not great." Id. On physical examination, Petitioner continued to have some decreased sensations. Id. at 104. Deep tendon reflexes were trace in his left patella and bilateral brachioradialis and biceps and absent otherwise. Id. Dr. Knappenberger's assessment was "[n]europathy" and noted Petitioner's history of GBS. Id. Dr. Knappenberger also noted Petitioner "continue[d] to have a modest but unchanging amount of sensory loss and he [] seem[ed] to have plateaued fairly quickly after his acute illness." Id. He ordered repeat EMG/NCS as well as bloodwork to rule out issues that may be compounding Petitioner's recovery. Id.

On March 10, 2017, Petitioner visited neurologist Dr. Daniel Rodriguez for a consultation regarding his improvement. Pet. Ex. 5 at 19-21. Dr. Rodriguez informed Petitioner "recovery could take place over a year or longer" and "he might have complete recovery or slightly less than complete recovery but [Dr. Rodriguez] expect[ed] him to be fully functional." Id. at 20.

A repeat EMG/NCS was done on April 6, 2017 and showed "electrodiagnostic evidence of a moderately severe generalized peripheral polyneuropathy[] with mixed axonal and demyelinating features." Pet. Ex. 4 at 77-80. Additionally, "[t]here [was] an apparent superimposed right ulnar mononeuropathy at the elbow." Id. at 77.

Petitioner returned to Dr. Rodriguez on June 12, 2017. Pet. Ex. 5 at 12-13. Petitioner reported "slight improvement" with "bothersome discomfort." Id. at 13. Dr. Rodriguez reviewed Petitioner's recent EMG/NCS. Id. Physical examination revealed absent reflexes. Id.

[19] During this time, Petitioner was experiencing back pain secondary to his gallbladder, making it difficult to differentiate "gallbladder back pain from other back pain." Pet. Ex. 2 at 101. He had a surgery scheduled to remove his gallbladder the following week for reasons unrelated to this vaccine claim. Id.

Assessment was GBS. Id. On Dr. Rodriguez's suggestion, Petitioner increased his gabapentin dosage from 400 mg every eight hours to 600 mg every eight hours. Id. As of 2020, Petitioner was on the maximum dosage of gabapentin, 3600 mg daily. See, e.g., Pet. Ex. 106 at 8, 15.

D. Petitioner's Affidavits and Declaration[20]

Petitioner averred that he received a Prevnar 13 vaccination on August 10, 2016. Pet. Ex. 8 at ¶ 2; Pet. Ex. 9 at ¶ 6. Approximately two weeks post-vaccination, around August 26 or 27, he developed neurological symptoms later diagnosed and treated as GBS. Pet. Ex. 8 at ¶ 3; Pet. Ex. 9 at ¶ 7; Pet. Ex. 160 at ¶ 4.

When he first sought medical attention at Fort Harrison, Petitioner stated that he told his provider he "had not had any recent illness, with no coughing, shortness of breath, nausea, vomiting, or irregular stools." Pet. Ex. 160 at ¶ 4.

Petitioner acknowledged, however, that during his hospitalization at Bozeman Deaconess Hospital, "the medical providers made notes that [he] had suffered from a diarrheal illness some period of weeks before," with some notations placing onset of a diarrheal illness one week before neurological symptoms or several weeks prior to neurological symptoms. Pet. Ex. 160 at ¶ 6. He maintained that these notations coincided with when his treating physicians "first suspected that a diarrheal illness had triggered [his] GBS," and "[t]hey later changed their opinion to believe that [his] [Prevnar 13] vaccination had been the trigger for [his] GBS." Id. at ¶ 7.

Additionally, because Deaconess is a teaching hospital, Petitioner suspected "[t]hey must have been using [him] to train the students" because of his rare condition. Pet. Ex. 160 at ¶ 8. He guessed that around 25 medical students and teachers inquired about whether he had experienced diarrhea. Id. Petitioner explained that he "could have said that [he] had some diarrhea in the recent past" due to the repeated questioning and "clouded" thinking as a result of prescribed opioid medication. Id. at ¶¶ 8-9. Looking back, Petitioner believed he "was likely referring to merely loose stools" he experienced due to his use of Metamucil fiber. Id. at ¶ 9. Petitioner maintained that he did not experience diarrheal symptoms consistent with *C. jejuni*, which he described as "severe and unmistakable, with cramping, abdominal pain, very loose or [] bloody stools, resulting [in] dehydration, and fever, all of which usually lasts a week."[21] Id. at ¶¶ 10-11.

Lastly, Petitioner maintained that providers at Bozeman Deaconess Hospital did not ask him whether he recently received any vaccinations. Pet. Ex. 160 at ¶ 11. But see, e.g., Pet. Ex. 6 at 24 (Dr. Knappenberger, on September 6, 2016, during Petitioner's hospitalization, documenting Petitioner received a Prevnar 13 vaccination "about [three] weeks" prior). Petitioner further stated that once he mentioned his recent Prevnar 13 vaccination to his neurologist Dr. Knappenberger, Dr. Knappenberger's "countenance immediately shifted," with "every record thereafter[] [] associat[ing] [his] GBS with [his] preceding [] vaccination as [the]

[20] Petitioner submitted two affidavits and one declaration. Pet. Exs. 8-9, 160.

[21] Petitioner does not appear to have any medical training.

cause." Pet. Ex. 160 at ¶ 12. Petitioner further asserted Dr. Knappenberger "made sure that [his] medical record[s] reflected that [he] should not receive [flu] vaccinations in the future, because of his strong belief that [Petitioner] had suffered from vaccine-related GBS." Id. But see Pet. Ex. 3 at 120 (Petitioner's PCP Dr. Swoboda writing the flu vaccine was deferred indefinitely).

E. Expert Reports[22]

1. Petitioner's Expert, Dr. Zurab Nadareishvili[23]

a. Background and Qualifications

Dr. Nadareishvili is board certified in neurology and vascular neurology. Pet. Ex. 100 at 1. He obtained an M.D. and Ph.D. and received neurology training in the Republic of Georgia before moving to the United States. Id. at 1-2. He completed an internal medicine internship and neurology residency at Georgetown University Hospital as well as a vascular neurology fellowship in stroke diagnostics and therapeutics at the National Institute of Health in Bethesda, Maryland. Id. at 2. Since 2011, Dr. Nadareishvili has held clinical and academic appointments in the DC area. Id. at 3-4. He is currently an associate professor of neurology at George Washington University and the medical director of the stroke program at Virginia Hospital Center. Id. at 1. Throughout his career, he has won various honors and awards, authored or co-authored over 30 peer-reviewed publications. Id. at 5-6, 11-14.

b. Opinion

Dr. Nadareishvili opined, "more likely than not," that Petitioner developed GBS 15 days post-Prevnar 13 vaccination and that the Prevnar 13 vaccination "was a substantial factor" in causing Petitioner's GBS. Pet. Ex. 99 at 1-2, 18; see also Pet. Ex. 101 at 6.

i. Althen Prong One[24]

Dr. Nadareishvili proposed that the Prevnar 13 vaccination can cause GBS via molecular mimicry. Pet. Ex. 99 at 7-11. He explained "molecular mimicry describes the immunological

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[22] Although the undersigned has reviewed all expert reports, for the sake of brevity, this Decision does not include every detail of the experts' opinions. Instead, the undersigned focuses on the experts' material opinions, as they relate to the relevant issue of causation.

[23] Dr. Nadareishvili provided two expert reports. Pet. Exs. 99, 101.

[24] The undersigned provides only a brief summary of Dr. Nadareishvili's opinions on Althen prong one as the undersigned's Decision does not turn on prong one. Further, Dr. Steinman provides a more robust theory. For the sake of brevity, this Decision does not discuss similar or duplicative opinions related to molecular mimicry by Dr. Nadareishvili that are discussed by Dr. Steinman. Nor does the undersigned address other theories posited by Dr. Nadareishvili, such as the "fertile field" model, as they are not germane to the undersigned's Decision.

processes of crossreactivity due to similarity of antigens between a host and a microorganism or components of microorganisms, such as those contained within a vaccine." Id. at 7.

Autoimmune responses are "highly individualized" due to interactions between environmental changes and host genetics. Pet. Ex. 99 at 9. According to Dr. Nadareishvili, "[g]enetic traits, or more precisely the expression of a genetic trait, remains a fundamental variable in the individual's maintenance of immunologic homeostasis, and this leads to a wide spectrum of variance in the susceptibility to autoimmune disease." Id. Thus, for molecular mimicry to lead to the development of autoimmune disorders like GBS, one must be "susceptible to a disruption of self-tolerance." Id. at 10.

More specifically, Dr. Nadareishvili opined Prevnar 13 vaccination is a "plausible trigger" for GBS. Pet. Ex. 99 at 10. He discussed the ingredients of the Prevnar 13 vaccine. Id. at 11, 13. And he explained that cross-reaction between the vaccine and self-antigens will still occur even if homology is not identified. Id. at 12-13. He also noted how Prevnar 13 "shared significant antigen resemblance with pneumococcal strains" and argued this is persuasive evidence in support of molecular mimicry. Pet. Ex. 101 at 5.

Overall, he mentioned two potential mechanisms that can lead to autoimmunity via molecular mimicry and concluded that "the Prevnar 13 vaccine can play a significant causal role in initiating/triggering" GBS. Pet. Ex. 99 at 15 (emphasis omitted); see also Pet. Ex. 101 at 5-6. The first, he explained, occurs when the Prevnar 13 vaccine is administered intramuscularly, and such injection produces local inflammation and recruitment of antigen presenting cells. Pet. Ex. 99 at 15. The "muscle[s] are richly innervated with twigs of peripheral nerve fibers, particular the muscle spindles are innervated by fibers enriched in GQ1b ganglioside, and these self-antigens can be presented in the context of a heightened immune response brought about by the adjuvants contained within the vaccine." Id. The second mechanism is "the potential for structural homology to lead to molecular mimicry, or even non-homologous molecular mimicry to occur," even though there is no known glycan sequence homology. Id.

Dr. Nadareishvili cited literature to support his opinion that the Prevnar 13 vaccine can cause GBS. See Pet. Ex. 99 at 12. In Haber et al.,[25] there were 11 reports of GBS after the Prevnar 13 vaccination,[26] one aged 19 to 64, and 10 in the age range of 65 and older. Pet. Ex. 32 at 4-5, 3 tbl.2a, 4 tbl.2b. Median onset was nine days post-vaccination and median patient age

[25] Penina Haber et al., Post-Licensure Surveillance of 13-Valent Pneumococcal Conjugate Vaccine (PCV13) in Adults Aged ≥19 Years Old in the United States, Vaccine Adverse Event Reporting System (VAERS), June 1, 2012–December 31, 2015, 34 Vaccine 6330 (2016). This article was also cited as Pet. Ex. 140, Resp. Ex. A, Tab 2, and Resp. Ex. C, Tab 21.

[26] One of the 11 patients also received a flu vaccine. Pet. Ex. 32 at 4.

was 68 years. Id. at 4. Cordonnier et al.[27] conducted a study to assess the immunogenicity and safety of pneumococcal vaccines (both Prevnar 13 and the 23-valent pneumococcal polysaccharide vaccine ("PPSV23")) administered in allogeneic hematopoietic stem cell transplant patients. Pet. Ex. 20 at 1-2. One patient developed GBS 29 days after the fourth dose of Prevnar 13 and one day after PPSV23. Id. at 7. The authors noted GBS was "possibly related to [] vaccination," but that the patients with severe adverse events, including the one patient who developed GBS, "had a complex constellation of comorbid conditions, received concomitant medications, and were exposed to multiple infections and [chronic graft-vs-host disease ("GVHD")]," which made "it [] difficult to establish a clear causal relationship between these events and [Prevnar 13]." Id. at 9.

Dr. Nadareishvili acknowledged the association between Prevnar 13 and GBS is rare and "has not been proven by epidemiologic studies." Pet. Ex. 99 at 12. He argued, however, that an absence of epidemiologic evidence "should not be interpreted to negate the biologic/medical plausibility of pneumococcal glycoconjugate vaccines as a causal trigger of GBS." Id.

He also noted case reports have indicated an association between Prevnar 13 and GBS. Pet. Ex. 99 at 15. However, he did not cite or discuss any of these case reports. See id.

In his supplemental expert report, Dr. Nadareishvili briefly discussed case reports of GBS post-pneumococcal infection to show molecular mimicry is the most likely pathologic mechanism here given the similarities he asserted were present between the Prevnar 13 vaccine and wild-virus pneumococcal infections. Pet. Ex. 101 at 4-5. Bianchi and Domenighetti[28] reported an association of bacteremic *Pneumococcus pneumoniae* infection and GBS in a 78-year-old man who developed GBS while hospitalized for an infection. Pet. Ex. 102 at 1. The authors hypothesized "the infection could induce . . . GBS" although this case "seem[ed] to be related partly to a simple fortuitous association." Id. at 2. They noted that molecular mimicry "has been postulated to explain the postinfectious, immunologic nature of [GBS]." Id.

White et al.[29] discussed a case of GBS post-*Streptococcus pneumoniae* ("*S. pneumoniae*") in a 68-year-old woman. Pet. Ex. 103 at 1. The authors noted that it was "possible" that molecular mimicry occurred in their patient. Id. at 3. Lastly, El Khatib et al.[30]

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[27] Catherine Cordonnier et al., Immunogenicity, Safety, and Tolerability of 13-Valent Pneumococcal Conjugate Vaccine Followed by 23-Valent Pneumococcal Polysaccharide Vaccine in Recipients of Allogeneic Hematopoietic Stem Cell Transplant Aged ≥2 Years: An Open-Label Study, 61 Clinical Infectious Diseases 313 (2015).

[28] Giorgia Bianchi & Guido Domenighetti, Pneumococcus Pneumoniae Infection and Guillain-Barré Syndrome: Fortuitous or Specific Association?, 32 Intensive Care Med. 338 (2006).

[29] B. White et al., A Novel Pneumococcus with a New Association, 9 Travel Med. Infectious Disease 84 (2011).

[30] Hassan El Khatib et al., Case Report: Guillain-Barre Syndrome with Pneumococcus – A New Association in Pediatrics, 11 IDCases 36 (2018).

presented a case of GBS in a 13-year-old male whose blood cultures were positive for *S. pneumoniae*. Pet. Ex. 104 at 1-2. His medical history noted he had not received any dose of a pneumococcal vaccine. Id. at 2. Like White et al., El Khatib et al. noted it was "possible" the infection caused GBS via molecular mimicry. Id.

ii. Althen Prongs Two and Three

Dr. Nadareishvili opined, to a reasonable degree of probability, Petitioner's Prevnar 13 vaccine acted as a substantial factor in causing/triggering his GBS 15 days thereafter. Pet. Ex. 99 at 15-16.

He noted diagnosis was not seriously questioned by any of Petitioner's treating physicians once he was diagnosed with GBS. Pet. Ex. 99 at 16. He opined Petitioner suffered specifically from AIDP[31] due to his EMG results.[32] Id.; Pet. Ex. 101 at 1-3.

Dr. Nadareishvili also agreed with the treating physicians who concluded Petitioner's Prevnar 13 vaccine "was the likely environmental trigger for his GBS." Pet. Ex. 99 at 16. He opined the temporal association of vaccination and onset (15 days) was "very strongly supportive of the vaccination as a cause." Pet. Ex. 99 at 17.

With regard to alternative causes, Dr. Nadareishvili opined that he "[could not] entirely rule out a diarrheal illness as a potential cause." Pet. Ex. 99 at 17; see also Pet. Ex. 101 at 6 ("I cannot entirely rule out the potential contributory role of a diarrheal illness as a concomitant significant factor."). He acknowledged that early medical records documented Petitioner's reports of a recent diarrheal illness "which could potentially have been causative." Pet. Ex. 99 at 17.

But even with the documented diarrheal illness, Dr. Nadareishvili maintained the Prevnar 13 vaccine was a "substantial factor" in Petitioner's GBS for two reasons. Pet. Ex. 99 at 17. First, Petitioner's clinical course resembled AIDP, not AMAN.[33] Id. He opined that AMAN is linked to *C. jejuni*, not AIDP. Id.

Second, Dr. Nadareishvili wrote "[i]ndigestion and loose stools are not evidence of such a [*C. jejuni*] infection [but] could represent another enteral virus or bacteria." Pet. Ex. 99 at 17. Dr. Nadareishvili did not cite to any literature to support this aspect of his opinion. See id.

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[31] "The most common subtype [of GBS] in North America and Europe, comprising more than 90 percent of cases, is [AIDP], which has the pathologic and electrodiagnostic features of focal demyelination of motor and sensory peripheral nerves and nerve roots." 42 C.F.R. § 100.3(c)(15)(ii).

[32] See supra note 4.

[33] Compared to AIDP, AMAN "is generally seen in other parts of the world and is predominated by axonal damage that primarily affects motor nerves. AMAN lacks features of demyelination." 42 C.F.R. § 100.3(c)(15)(ii).

However, Petitioner's literature is replete with statements and findings that many GBS patients report GI or diarrheal illnesses prior to onset. See, e.g., Pet. Ex. 35 at 2; Pet. Ex. 38 at 2-4, 4 fig.1; Pet. Ex. 61 at 3; Pet. Ex. 89 at 2.[34] In Koga et al., for example, the authors examined antecedent symptoms in GBS and found 29% (51/176) of their patients had a GI illness prior to onset of GBS and 27% (47/176) reported diarrhea. Pet. Ex. 38 at 2-4, 4 fig.1; see also Guillain-Barré Syndrome, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=110689 (last visited Feb. 7, 2025) (acknowledging that GBS is "frequently seen after an enteric or respiratory infection").

In response to Dr. Chaudhry's opinion that Petitioner developed GBS secondary to a diarrheal illness, Dr. Nadareishvili disagreed, noting that there was no diagnostic testing done to confirm a diarrheal illness. Pet. Ex. 101 at 1. He also asserted that Dr. Chaudhry failed to acknowledge treating physician references to vaccination as the cause of Petitioner's GBS. Id. at 3 (citing Dr. Knappenberger's diagnosis of "GBS following vaccination" on September 21, 2016 (quoting Pet. Ex. 2 at 22)).

Dr. Nadareishvili concluded that "more likely than not," Petitioner's Prevnar 13 vaccination was a substantial factor in causing his GBS. Pet. Ex. 99 at 17.

2. Petitioner's Expert, Dr. Lawrence Steinman[35]

a. Background and Qualifications

Dr. Steinman is board certified in neurology and has practiced neurology at Stanford University for over 40 years. Pet. Ex. 112 at 1; Pet. Ex. 113 at 1-2. He received his B.A. from Dartmouth College in 1968 and his M.D. from Harvard University in 1973. Pet. Ex. 113 at 1. Thereafter, he completed a surgery internship, pediatrics residency, and pediatric and adult neurology residency at Stanford University Hospital, as well as three fellowships, including one in clinical immunology. Id. Dr. Steinman is currently a Professor at Stanford University. Id. Dr. Steinman "is actively involved in patient care" and "ha[s] cared for hundreds of adults and children with various forms of inflammatory neuropathy, [GBS], transverse myelitis, [ADEM], neuromyelitis optica[,] and [MS]." Pet. Ex. 112 at 1. He has authored or co-authored over 600 publications. Pet. Ex. 113 at 5-50. Dr. Steinman has authored papers on molecular mimicry, as demonstrated by his CV. See id. One of Dr. Steinman's specialties is in the area of MS, and he has received a Charcot Prize for Lifetime Achievement due to his research in MS. Pet. Ex. 112 at 2. In 2015, he was elected to the National Academy of Sciences. Id.

b. Opinion

Dr. Steinman opined "the Prevnar 13 vaccine, more likely than not, triggered [GBS] in [] Petitioner. The diarrheal infection of unknown microbiologic characterization [was] also

[34] All of this literature was filed before Dr. Nadareishvili's expert reports and reviewed by Dr. Nadareishvili in preparing said reports. See Pet. Ex. 99 at 1.

[35] Dr. Steinman provided two expert reports. Pet. Exs. 112, 142.

evidence of another substantial factor that could contribute to the triggering of GBS." Pet. Ex. 112 at 31; Pet. Ex. 142 at 29.

i. Althen Prong One[36]

Dr. Steinman opined that the Prevnar 13 vaccination can cause GBS. Pet. Ex. 112 at 1, 31. Dr. Steinman reviewed the components of the vaccine and the main targets of the human immune response in GBS and proposed two mechanisms whereby molecular mimicry can trigger GBS following Prevnar 13 vaccination. The first involves homology between the components in the vaccine and phosphoglycerol components in the myelin and axons of peripheral nerves. Id. at 6-22. The second involves homology between CRM_{197} in the vaccine and Contactin-1, a protein found in humans. Id. at 22-30.

1. Phosphoglycerol[37] in Serotypes 18C and 23F

The first mechanism described by Dr. Steinman involves homology between phosphoglycerol in the Prevnar 13 vaccine, present in the antigens of *S. pneumoniae* serotypes 18C and 23F, and phospholipids in the human myelin sheath. Pet. Ex. 112 at 6-22; Pet. Ex. 142 at 1-19; see also Pet. Ex. 120 at 24 (Prevnar 13 package insert).

Dr. Steinman cited evidence, including the vaccine patent and various studies, to explain this theory. Pet. Ex. 112 at 6-17; see also Pet. Ex. 142 at 1-19. He opined that phospholipids[38] are the targets of antibodies in GBS. Pet. Ex. 112 at 9. He asserted that antibodies to phosphoglycerol structures interact with myelin components triggering GBS. Id. at 9-10. Based on his own research, Dr. Steinman explained that "phospholipids are components of the myelin sheath in humans, and [] they are targeted by antibodies" leading to neuroinflammation in GBS. Id. at 9; see, e.g., Pet. Ex. 118 (showing autoantibodies primarily target a phosphoglycerol

[36] The undersigned provides only a brief summary of Dr. Steinman's opinions on Althen prong one as she is familiar with his theory and this case does not turn on prong one.

[37] Phospho- is a "prefix [] indicating the presence of phosphorus in a compound." Stedman's Medical Dictionary 1486 (28th ed. 2006). Glycerol is "[a] sweet viscous fluid obtained by the saponification of fats and fixed oils; used as a solvent, as a skin emollient, . . . and as a vehicle and sweetening agent." Stedman's at 820.

[38] Phospholipid is defined as "any lipid that contains phosphorus, including those with a glycerol backbone (phosphoglycerides and plasmalogens) . . . . Phospholipids are the major form of lipid in all cell membranes." Phospholipid, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=38759 (last visited Feb. 7, 2025).

component of myelin in MS);[39] Pet. Ex. 121 at 5 ("In our study we detected a wide range of anti-phospholipid antibodies in patients with idiopathic GBS. All nine GBS patients developed anti-phospholipid antibodies directed against at least one lipid during the course of the disease . . . .").[40] Of note, Nakos et al. concluded,

> [i]t is not well understood whether these anti-phospholipid antibodies play a role in the pathogenesis of the polyneuropathy or represent a part of a more extensive immunoreaction that takes place in [] GBS. However, immunopathology in autopsies suggests that antibody mediated injury is a predominant disorder in the demyelinating form of GBS. The immune attack is directed against components of Schwann cell[41] membrane and is accompanied by the characteristic feature of vesicular demyelination. Therefore, it is crucial to investigate how anti-phospholipid antibodies are related to specific antigens in Schwann cell membrane.
>
> . . . .
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> Our findings suggest that in GBS there is a more extensive immune reaction, beyond the well known antiganglioside production, which has been related to the demyelination of the peripheral nerves.

Pet. Ex. 121 at 6-7.

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[39] Peggy P. Ho et al., Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation, 4 Sci. Translational Med. 1 (2012). Dr. Steinman is a named author. Ho et al. examined the role of lipids in autoimmune demyelination, specifically "whether lipids in the myelin sheath are targeted by autoimmune responses in MS." Pet. Ex. 118 at 1, 9. They found "myelin phospholipids are targeted by autoimmune responses in MS and that these myelin phospholipids represent a natural anti-inflammatory class of compounds that have potential as therapeutics for MS." Id. at 9.

[40] G. Nakos et al., Anti-Phospholipid Antibodies in Serum from Patients with Guillain-Barré Syndrome, 31 Intensive Care Med. 1401 (2005).

[41] Schwann cells are "any of the large nucleated cells whose cell membrane spirally enwraps the axons of myelinated peripheral neurons and is the source of myelin; a single Schwann cell supplies the myelin sheath between two nodes of Ranvier." Schwann Cell, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=64407 (last visited Feb. 7, 2025). The nodes of Ranvier are "constrictions occurring on myelinated nerve fibers at regular intervals of about [one] mm; at these sites the myelin sheath is absent and the axon is enclosed only by Schwann cell processes." Nodes of Ranvier, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=93095 (last visited Feb. 7, 2025).

Dr. Steinman also discussed a study by Bryson et al.[42] of antibodies directed to serotype 23F from humans who had received PPSV23, which also contains serotype 23F.[43] Pet. Ex. 112 at 17-21 (citing Pet. Ex. 128). He explained Bryson et al. used X-rays to "demonstrate[] that the immune response to *S. pneumoniae* serotype 23F after [PPSV23] targets the phosphoglycerol in the polysaccharide capsule of serotype 23F." Id. at 17 (citing Pet. Ex. 128). Dr. Steinman opined the X-rays in Bryson et al. showed human antibodies targeting serotype 23F, "demonstrat[ing] that the immune response to the phosphoglycerol in the polysaccharide capsule of serotype 23F is critical to the human immune response to serotype 23F." Id. at 19-20. Dr. Steinman further opined the "data from the Bryson [et al.] article demonstrate unequivocally that the immune response to the serotype 23F component of [PPSV23] targets the phosphoglycerol in serotype 23F." Id. at 21 (emphasis omitted). "Since the 23F and 18C components of Prevnar 13 also contain the phosphoglycerol moiety targeted by the antibodies generated by [PPSV23], it is very likely that the immune response to 23F and 18C components of Prevnar 13 vaccine also targets the phosphoglycerol moiety." Id. at 21-22.

In summary, Dr. Steinman's first theory is based on molecular mimicry, and he opined antibodies to the phosphoglycerol structures in Prevnar 13 (via serotypes 18C and 23F) target an immune response in phospholipids in the myelin of peripheral nerves, triggering GBS. Pet. Ex. 112 at 9-22.

2. CRM_{197} and Contactin-1

The second homology posited by Dr. Steinman is between the protein carrier in the vaccine, CRM_{197},[44] and Contactin-1,[45] targeted in some cases of GBS. Pet. Ex. 112 at 22-30. Prevnar 13 is a conjugate vaccine in which the individual polysaccharides of the capsular antigens of *S. pneumoniae* are linked to a non-toxic diphtheria CRM_{197} protein. Id. at 6 (citing Pet. Ex. 120 at 24). "CRM_{197} is a nontoxic variant of diphtheria toxin," used as a protein carrier which makes the vaccine more immunogenic. Id. at 6, 22 (quoting Pet. Ex. 120 at 24). CRM_{197}

[42] Steve Bryson et al., Structures of Preferred Human IgV Genes–Based Protective Antibodies Identify How Conserved Residues Contact Diverse Antigens and Assign Source of Specificity to CDR3 Loop Variation, 196 J. Immunology 4723 (2016).

[43] Serotype 23F is included in both Prevnar 13 and PPSV23. Pet. Ex. 112 at 17; see Pet. Ex. 120 (Prevnar 13 package insert); Pet. Ex. 130 (PPSV23 package insert).

[44] Protein carrier "CRM_{197} is a nontoxic variant of diphtheria toxin isolated from cultures of *Corynebacterium diphtheriae* strain C7 (β197) grown in a casamino acids and yeast extract-based medium." Pet. Ex. 120 at 24 (Prevnar 13 package insert).

[45] Contactin-1, or CNTN1, "is a key axonal adhesion molecule, which interacts with CNTNAP1 (previously known as Caspr1) on the axon and neurofascin-155 on the glial side, and is essential for the formation of the paranodal septate-like junction." Pet. Ex. 133 at 2 (Yumako Miura et al., Contactin I IgG4 Associates to Chronic Inflammatory Demyelinating Polyneuropathy with Sensory Ataxia, 138 Brain 1484 (2015)).

differs from diphtheria toxin by only one amino acid, and is therefore not toxic. Id. at 6, 22-23; see also Pet. Ex. 132 at 1.[46]

Again, based on his own research, Dr. Steinman determined that molecular mimicry may occur between CRM_{197} and Contactin-1, a molecule that has been identified in patients with GBS. Pet. Ex. 112 at 23. Dr. Steinman relied on Miura et al., a study done on patients with chronic inflammatory demyelinating polyneuropathy ("CIDP"). Id. (citing Pet. Ex. 133). Miura et al. focused their research on patients with CIDP, but used sera from patients with GBS, MS, and healthy patients as controls. Pet. Ex. 133 at 2. They found that five of the 200 patients with GBS had anti-Contactin-1 Immunoglobulin G ("IgG") antibodies. Id. at 3, 6 tbl.2.

Miura et al. explained the theory of pathogenesis relevant to Dr. Steinman's theory, as it relates to Contactin-1:

> Cell adhesion molecules play a crucial role in the formation of the nodes of Ranvier and in the rapid propagation of the nerve impulses along myelinated axons. In the peripheral nerves, the domain organization of myelinated axons depends on specific axo-glial contacts between the axonal membrane and Schwann cells at nodes, paranodes[,] and juxtaparanodes.

Pet. Ex. 133 at 2.

Miura et al. identified Contactin-1 (CNTN1) as one of the targets of autoantibodies in some patients with GBS. Pet. Ex. 133 at 3. Other articles provided by Petitioner also note antibodies to paranodal proteins are found in GBS. See, e.g., Pet. Ex. 152 at 1, 6 (showing nodal proteins are targets in GBS and that Contactin is an "immune target[] of autoantibodies" in GBS);[47] Pet. Ex. 153 at 3 fig.1. (illustrating the structure of the peripheral nervous system nodes, paranodes, and Contactin (CNTN1 and CNTN2));[48] Pet. Ex. 158 at 3 ("Antibodies to paranodal proteins are found in MS and in both [GBS] and chronic inflammatory polyneuropathy.").[49]

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[46] Michael Bröker et al., Biochemical and Biological Characteristics of Cross-Reacting Material 197 (CRM_{197}), a Non-Toxic Mutant of Diphtheria Toxin: Use as a Conjugation Protein in Vaccines and Other Potential Clinical Applications, 39 Biologicals 195 (2011).

[47] Jérôme J. Devaux et al., Nodal Proteins Are Target Antigens in Guillain-Barré Syndrome, 17 J. Peripheral Nervous Sys. 62 (2012).

[48] Janev Fehmi et al., Nodes, Paranodes and Neuropathies, 89 J. Neurology Neurosurgery & Psychiatry 61 (2018).

[49] Tobias V. Lanz et al., Roadmap for Understanding Mechanisms on How Epstein-Barr Virus Triggers Multiple Sclerosis and for Translating These Discoveries in Clinical Trials, 12 Clinical & Translational Immunology e1438 (2023). Dr. Steinman is a named author.

Based on this information, Dr. Steinman conducted a BLAST[50] search to determine whether there was homology between CRM_{197} and Contactin-1.[51] Pet. Ex. 112 at 24. He found a sequence[52] ("WEQ sequence") that "might be capable of inducing a neuroinflammatory disease." Id. at 27. He found "it is an epitope in diphtheria toxin, which provides the basis for CRM_{197}." Id. at 28. In addition to the WEQ sequence, Dr. Steinman identified another sequence[53] that "has known cross-reactivity with epitopes described in humans and on the [*Corynebacterium diphtheriae*] microbe that is the basis for CRM_{197}." Id.

Dr. Steinman opined these sequences were significant due to five identical amino acids in a nervous system protein. Pet. Ex. 112 at 24. He cited a number of papers, including some that he authored or co-authored, to support his opinion that homology of just five amino acids can induce an immune response consistent with his theory here. Id. For example, he cited the Gautam et al.[54] studies for the proposition that autoimmune encephalomyelitis could be induced with homology at five amino acids. Id. (citing Pet. Exs. 134-36).

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[50] A BLAST (Basic Local Alignment Search Tool) search "finds regions of similarity between biological sequences. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance." BLAST, https://blast.ncbi.nlm.nih.gov/Blast.cgi (last visited Feb. 7, 2025).

[51] For a complete explanation of Dr. Steinman's investigation, including his discussion on the number of amino acids required for homology relevant to molecular mimicry as well as the procedure he followed in conducting his BLAST searches and his research using the Immune Epitope Database ("IEDB"), see Pet. Ex. 112 at 24-29. Based on his IEDB search, Dr. Steinman was referred to a paper by Raju et al. that reported a human immune response to the diphtheria toxin and identified the WEQAKALSVE sequence. Pet. Ex. 112 at 28-29 (citing Pet. Ex. 139 at 2 (Raghavanpillai Raju et al., Epitopes for Human CD4+ Cells on Diphtheria Toxin: Structural Features of Sequence Segments Forming Epitopes Recognized by Most Subjects, 25 Euro. J. Immunology 3207 (1995))). For Dr. Steinman's responses to Dr. Whitton's criticisms regarding his BLAST search and the Silvanovich et al. criteria, see Pet. Ex. 142 at 20-26, 28-29; Pet. Ex. 138 (Andre Silvanovich et al., The Value of Short Amino Acid Sequence Matches for Prediction of Protein Allergenicity, 90 Toxicological Scis. 252 (2006)).

[52] The sequence is "WEQAKALSVE," which "has five of ten identical amino acids." Pet. Ex. 112 at 27.

[53] The second sequence is "EYMAQACAGNRVRR." Pet. Ex. 112 at 28.

[54] Anand M. Gautam et al., A Polyalanine Peptide with Only Five Native Myelin Basic Protein Residues Induces Autoimmune Encephalomyelitis, 176 J. Experimental Med. 605 (1992); Anand M. Gautam et al., Minimum Structural Requirements for Peptide Presentation by Major Histocompatibility Complex Class II Molecules: Implications in Induction of Autoimmunity, 91 Immunology 767 (1994); Anand M. Gautam et al., A Viral Peptide with Limited Homology to a Self Peptide Can Induce Clinical Signs of Experimental Autoimmune Encephalomyelitis, 161 J. Immunology 60 (1998). Dr. Steinman is a named author in all of these papers.

In summary, Dr. Steinman averred that "[this] theory provides actual detailed data for molecular mimics in [] $CRM_{[197]}$ in the Prevnar 13 vaccine" and "shows how these mimics could trigger inflammatory neuropathy culminating in GBS." Pet. Ex. 112 at 29 (emphasis omitted).

ii. Althen Prong Two

Dr. Steinman opined that given the evidence he presented in his two molecular mimicry theories, "there is a logical basis for a cause and effect relationship between Prevnar 13[] and Petitioner's GBS." Pet. Ex. 112 at 30. He agreed with the diagnosis of GBS. Id. at 1.

Dr. Steinman acknowledged "[t]here was a diarrheal illness prior to the Prevnar 13 vaccine." Pet. Ex. 112 at 1 (citing Pet. Ex. 6 at 4). However, he assigned a lower weight to Petitioner's diarrheal illness as the trigger of Petitioner's GBS because a microbiologic diagnosis was not made. Id. at 30-31. "Had a microbiologic diagnosis of *C. jejuni* been ascertained, [he] would [have] place[d] such an illness as more likely than Prevnar 13. As an unknown diarrheal illness, [he] place[d] the illness lower." Id. at 31. Yet, he concluded the diarrheal illness was "evidence of another substantial factor that could contribute to the triggering of GBS." Pet. Ex. 142 at 29.

Overall, he opined that both the Prevnar 13 vaccine and diarrheal illness were "substantial factors" in Petitioner's development of GBS. Pet. Ex. 112 at 5, 30.

iii. Althen Prong Three

Dr. Steinman opined that Petitioner's onset of GBS was approximately 15 days after vaccination, which was "certainly consistent" with the timing known for GBS post-vaccination and the timing in Haber et al. and Schonberger et al. Pet. Ex. 112 at 1, 30 (citing Pet. Ex. 3 at 151; Pet. Ex. 32; Pet. Ex. 59).[55]

The 11 reports of possible GBS post-Prevnar 13 vaccination discussed in Haber et al. had a median onset of nine days post-vaccination. Pet. Ex. 32 at 4. The one report of a patient who developed GBS following Prevnar 13 and flu vaccinations was diagnosed with GBS 11 days post-vaccination. Id. Schonberger et al. examined cases of GBS following the 1976 swine flu vaccine and determined the period of increased risk of GBS following vaccination was within five weeks of vaccination, with a peak and two to three weeks. Pet. Ex. 59 at 1, 6.

Dr. Steinman concluded that the temporal relationship criteria of Althen prong three was fulfilled based on this interval. Pet. Ex. 112 at 31; Pet. Ex. 142 at 30.

[55] Lawrence B. Schonberger et al., Guillain Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977, 110 Am J. Epidemiology 105 (1979). This article is also cited as Pet. Ex. 141.

3. Respondent's Expert, Dr. Vinay Chaudhry[56]

a. Background and Qualifications

Dr. Chaudhry is board certified in neurology, neuromuscular diseases, electrodiagnostic medicine, and clinical neurophysiology. Resp. Ex. A at 1. He specializes in the field of neuromuscular diseases, including the management of peripheral neuropathies, and electrodiagnostic studies. Id. He received his M.B. and B.S. in India and then completed an internship and various residencies and fellowships from 1980 to 1989. Resp. Ex. B at 1-2. Thereafter, he began teaching neurology courses at Johns Hopkins University School of Medicine until 2021, when he moved to University of North Carolina, Chapel Hill School of Medicine. Id. at 2-3. Dr. Chaudhry has an active clinical practice where he sees over 2,000 patients per year, with most related to peripheral nerve diseases. Resp. Ex. A at 1. He has authored or co-authored over 200 publications. Resp. Ex. B at 5-22. "[He] [is] considered an expert in evaluation and treatment of patients with peripheral neuropathies including [GBS]." Resp. Ex. A at 1.

b. Opinion

Dr. Chaudhry agreed Petitioner's clinical course supports a diagnosis of GBS. Resp. Ex. A at 6-7. Contrary to Petitioner's experts, he opined that Petitioner's GBS was "more likely than not" caused by his diarrheal illness. Id. at 7, 12. He further opined "[t]here is no evidence to support that [Petitioner's] Prevnar 13 vaccine caused his GBS." Id. at 12.

He explained that GBS is a post-infectious immune disorder, with two-thirds of patients experiencing a GI or respiratory illness weeks prior to symptom onset. Resp. Ex. A at 7-8 (citing Resp. Ex. A, Tab 1 at 1-2 ("Two-thirds of adult patients report preceding symptoms of a respiratory or [GI] tract infection within [four] weeks of onset of weakness."); Resp. Ex. A, Tab 6 at 1, 6-7 (noting "*Campylobacter* infection is an important cause of acute diarrhea worldwide")). Because Petitioner had a diarrheal illness two to four weeks prior to the onset of his GBS, Dr. Chaudhry opined it was a "more likely" cause of Petitioner's GBS than vaccination. Id.

In support, he noted several treating physicians documented Petitioner's history of diarrheal illness. Resp. Ex. A at 7. For example, on September 3, 2016, emergency physician Dr. Cohen wrote Petitioner reported "diarrhea several weeks ago." Id. (quoting Pet. Ex. 6 at 5). Later that day, hospitalist Dr. Katz transcribed Petitioner reported a "brief diarrheal illness" over two weeks prior. Id. (quoting Pet. Ex. 6 at 20). Dr. Katz concluded that "[g]iven recent history of a diarrheal illness," and Petitioner's symptoms, Petitioner had GBS. Id. (quoting Pet. Ex. 6 at 23). And neurologist Dr. Knappenberger, on September 6, recorded that "about a month ago," Petitioner "developed a mild GI illness with diarrhea for a couple of days." Id. (quoting Pet. Ex. 6 at 24).

[56] Dr. Chaudhry submitted one expert report. Resp. Ex. A.

Dr. Chaudhry concluded that Petitioner's clinical course, notably his diarrheal illness two to four weeks prior to onset or neurologic symptoms, was a "rather typical antecedent history" seen in GBS patients. Resp. Ex. A at 7. Additionally, he asserted that most patients who develop GBS post-GI illness report a self-limited diarrheal illness. Id. at 12.

Dr. Chaudhry also provided reasons why he opined the Prevnar 13 vaccine does not cause GBS. Resp. Ex. A at 7-9. He first discussed Haber et al., which was cited by Petitioner's experts, and noted that although 11 cases of GBS had an onset within 42 days, the rate of GBS cases was not reported more often than would be expected. Id. at 7 (citing Pet. Ex. 32 at 1-2, 5 ("Our data mining analysis noted no disproportionate reporting for GBS.")).

He also cited Baxter et al.,[57] which reviewed GBS cases after vaccinations using Kaiser Permanente data from 1994 through October 2006 and noted that there was no increased risk of GBS observed. Resp. Ex. A at 8 (citing Resp. Ex. A, Tab 3 at 1-2). However, Prevnar 13 was not included in this study as it was not yet approved for use in adults. See Resp. Ex. A, Tab 3 at 1, 4; Pet. Ex. 32 at 1.

With regard to the theory of molecular mimicry, Dr. Chaudhry does not dispute the theory generally. See Resp. Ex. A at 8. He cited an article from Yuki,[58] who explained that the theory of "[m]olecular mimicry has been proposed to be a pathogenic mechanism of autoimmune disease . . . based on epidemiological, clinical, and experimental evidence of the association of infectious agents with autoimmune diseases and an observed cross-reactivity of antibodies raised by microbial components with host 'self' antigens." Resp. Ex. A, Tab 7 at 1.

Dr. Chaudhry disagreed that molecular mimicry was the applicable immune mechanism at play here because Petitioner did not have AMAN or Miller-Fisher GBS variant,[59] which are the only forms of GBS that have been associated with molecular mimicry. Resp. Ex. A at 8. Of note, those forms of GBS have been seen in the context of *C. jejuni*. Id. Dr. Chaudhry agreed that Petitioner was described as having an "axonal variant," although he was unable to verify this based on available information in the record. Id. at 7 (citing Pet. Ex. 4 at 77 (noting EMG showed evidence of "mixed axonal and demyelinating features"); Pet. Ex. 5 at 12 (noting Petitioner was assessed with "sensorimotor neuropathy . . . most likely a variant of [GBS]")).

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[57] Roger Baxter et al., Lack of Association of Guillain-Barré Syndrome with Vaccinations, 57 Clinical Infectious Diseases 197 (2013). This article was also cited as Resp. Ex. C, Tab 22.

[58] Nobuhiro Yuki, Ganglioside Mimicry and Peripheral Nerve Disease, 35 Muscle & Nerve 691 (2007).

[59] Miller-Fisher "is a subtype of GBS characterized by ataxia, areflexia, and ophthalmoplegia, and overlap between [Miller-Fisher] and AIDP may be seen with limb weakness." 42 C.F.R. § 100.3(c)(15)(iii).

4. Respondent's Expert, Dr. J. Lindsay Whitton[60]

a. Background and Qualifications

Dr. Whitton received his B.Sc. in molecular biology, his M.B., Ch.B. in medicine, and his Ph.D. in herpesvirus transcription from the University of Glasgow in Scotland in 1984. Resp. Ex. D at 1. Beginning in 1984, he joined Scripps Research Institute where he taught and conducted research. Id.; Resp. Ex. C at 1-2. He "studied (and published on) viral pathogenesis, and the immune responses to virus infections and to vaccines," and he has "published on both the adaptive and innate immune responses[] and on molecular mimicry." Resp. Ex. C at 2. Dr. Whitton is a member of various professional societies and editorial boards and has authored or co-authored approximately 200 publications. Resp. Ex. D at 1-15. Dr. Whitton does not hold a medical license, provide patient care, or diagnose or treat patients with GBS. Resp. Ex. C at 3.

b. Opinion

Dr. Whitton concluded Petitioner's Prevnar 13 vaccine did not play a part in causing his alleged injury. Resp. Ex. F at 15.

Dr. Whitton did not offer an opinion as to Petitioner's diagnosis or take a position on whether the diagnosis of GBS was appropriate. Resp. Ex. C at 3. His focus was on the question of whether the Prevnar 13 vaccine can cause GBS.[61] See Resp. Exs. C, E-F.

i. Althen Prong One

In his reports, Dr. Whitton described the Prevnar 13 vaccine, including its ingredients and the infection/disease against which it protects. Resp. Ex. C at 3-6. He also summarized features of GBS. Id. at 7-8.

Of note, Dr. Whitton, like Dr. Chaudhry, wrote "[two-thirds] of GBS cases are preceded by signs and/or symptoms of an infection, often of the respiratory or GI tracts." Resp. Ex. C at 7. These signs and symptoms of infection generally precede onset of GBS by one to four weeks, with a mean latency of one to two weeks for a GI infection. Id. (citing Resp. Ex. C, Tab 12 at 3

[60] Dr. Whitton submitted three expert reports. Resp. Exs. C, E-F.

[61] Dr. Whitton's criticisms of Dr. Nadareishvili's and Dr. Steinman's expert opinions are far ranging. See Resp. Exs. C, E-F. For the sake of brevity and clarity, the undersigned attempts to discuss the material points and omit discussion of criticism that is collateral to the central issues. And as noted above, the undersigned only briefly discusses Althen prong one as this case does not turn on it. Thus, many of Dr. Whitton's points have not been discussed here. However, the undersigned has reviewed Dr. Whitton's reports and the medical literature in its entirety and has taken all of this evidence into consideration in reaching her opinions.

fig., 4).[62] Although testing for infectious agents in GBS cases are routinely conducted, studies have shown that specific infectious agents are identified in only half of GBS patients. Id. (citing Resp. Ex. C, Tab 11 at 3 tbl.1 (documenting an infectious agent was identified in 87 of 154 patients, or 56% of patients)).[63]

With regard to infections as triggers of GBS, Dr. Whitton discussed *C. jejuni* and noted it is the most common infectious organism associated with the development of GBS. Resp. Ex. C at 8-9. He also noted other infectious organisms can be associated with GBS. Id. at 9. However, he asserted *S. pneumoniae*, which Prevnar 13 protects against, is not generally implicated as a cause of GBS. Id. He agreed that there are many infections associated with GBS, but *S. pneumoniae* is not known to be one of them. Id. at 9, 16. And since the organism does not trigger GBS, he argued the vaccine with the same antigens cannot do so. Id. at 10; Resp. Ex. E at 2.

Next, Dr. Whitton discussed the safety of Prevnar 13, explaining studies (Haber et al., Baxter et al., and Tseng et al.)[64] have not found an increased risk of GBS following pneumococcal vaccination. Resp. Ex. C at 10-11 (citing Pet. Ex. 32; Resp. Ex. C, Tab 3); Resp. Ex. E at 2-3 (citing Resp. Ex. E, Tab 1). To briefly summarize, Haber et al. reported 11 cases of GBS, and in ten of those patients, the Prevnar 13 vaccine was the only vaccine administered. Pet. Ex. 32 at 5. While the authors concluded that there was no disproportionate reporting for GBS, their reliance on VAERS data raised issues about the reliability of the results. Id. at 5-6. As noted by the authors, the limitations of VAERS "may include underreporting, varying quality of reports . . . , and the lack of an unvaccinated comparison group." Id. at 6.

Baxter et al. did not examine any GBS patients who received the Prevnar 13 vaccine because, as Dr. Whitton acknowledged, Prevnar 13 was not included in the study. Resp. Ex. A, Tab 3 at 1-2, 5; Resp. Ex. C at 11; see also Pet. Ex. 32 at 1 (noting Prevnar 13 was approved for use in adults in 2011, more than five years after Baxter et al. concluded its data collection). Two patients in Baxter et al. received PPSV23. Resp. Ex. A, Tab 3 at 5. The authors acknowledged that the study had "limited power to fully assess the risk of GBS following vaccination due to the rarity of the outcome." Id. at 8.

The study by Tseng et al. included adults 65 or older, and adverse events were compared between Prevnar 13 and PPSV23 instead of to a control group. Resp. Ex. E, Tab 1 at 1. Twelve cases of GBS were found, four in the Prevnar 13 group, and eight in the PPSV23 group. Id. at 6 tbl.3. The authors concluded there was "no significantly elevated risk" of GBS between the two

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[62] J.B. Winer et al., A Prospective Study of Acute Idiopathic Neuropathy. II. Antecedent Events, 51 J. Neurology Neurosurgery & Psychiatry 613 (1988).

[63] B.C. Jacobs et al., The Spectrum of Antecedent Infections in Guillain-Barré Syndrome: A Case-Control Study, 51 Neurology 1110 (1998).

[64] Hung Fu Tseng et al., Pneumococcal Conjugate Vaccine Safety in Elderly Adults, 5 Open Forum Infectious Diseases 1 (2018).

groups. Id. at 7. It is difficult to discern whether the combined incidence of GBS after these vaccinations was higher than the background rate for GBS.

While Dr. Whitton agreed that molecular mimicry is "real," he believed "it is very difficult to trigger disease via molecular mimicry." Resp. Ex. C at 12. For support he cited various articles, including the 2012 Institute of Medicine ("IOM") Report,[65] which stated that "[w]hile molecular mimicry is a well-established mechanism in selected animal models, its relevance to human autoimmune disease remains in most cases to be convincingly proven." Id. (quoting Resp. Ex. C, Tab 24 at 15).

After commenting on molecular mimicry generally, Dr. Whitton addressed the theories offered by Dr. Steinman. To summarize, Dr. Whitton argued reviews do not mention phospholipids as targets, and in fact, gangliosides are the targets in GBS.[66] Resp. Ex. E at 5-6. And for various reasons, Dr. Steinman's phosphoglycerol theory is speculative and his literature did not support his posited theory. Id. at 6-15; Resp. Ex. F at 1-7. Additionally, Dr. Whitton took issue with Dr. Steinman's CRM_{197} and Contactin-1 theory, including his use of a BLAST search, his "filter funnel" arguments, and his supportive literature.[67] Resp. Ex. E at 15-29; Resp. Ex. F at 7-15.

In summary, Dr. Whitton opined that Dr. Steinman presented "very little evidence" to support the Prevnar 13 vaccine, more likely than not, caused or contributed to Petitioner's development of GBS via molecular mimicry. Resp. Ex. E at 29. He noted that "Prevnar 13 has an excellent safety record" and he is "not aware of any reliable evidence that associates it with GBS." Id. He also noted *S. pneumoniae* is not causally associated with GBS, and thus, the same vaccine antigens cannot be associated with GBS. Id. Lastly, he opined that Dr. Steinman's molecular mimicry theories are speculative and lack evidence. Id. at 29-30; see also Resp. Ex. F at 16.

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[65] Inst. of Med., Evaluating Biological Mechanisms of Adverse Events, in Adverse Effects of Vaccines: Evidence and Causality 57 (Kathleen Stratton et al. eds., 2012).

[66] For example, he cited a review article from Hughes et al., which does not mention the word "phospholipids" as an important target in GBS. Resp. Ex. E at 5 (citing Resp. Ex. E, Tab 3 (Richard A.C. Hughes et al., Guillain-Barré Syndrome in the 100 Years Since Its Description by Guillain, Barré and Strohl, 139 Brain 3041 (2016))). And he cited Kanter et al., a paper he argued stated "gangliosides (not phospholipids) are the important target in GBS." Id. (citing Resp. Ex. E, Tab 2 at 1 ("Autoimmune responses directed against phospholipids and gangliosides contribute to the pathogenesis in systemic lupus erythematosus and [GBS], respectively.") (Jennifer L. Kanter et al., Lipid Microarrays Identify Key Mediators of Autoimmune Brain Inflammation, 12 Nature Med. 138 (2006) (Dr. Steinman is a named author))).

[67] For Dr. Whitton's arguments regarding the Expect value, or E-value, and Silvanovich et al., as well as the "filter funnel" approach, see Resp. Ex. E at 18-26.

ii. Althen Prongs Two and Three

Dr. Whitton noted Petitioner suffered from a diarrheal illness a few weeks prior to developing GBS, as discussed by Dr. Chaudhry. Resp. Ex. C at 7. He opined that because "GI infections are known to trigger GBS[] [and] Prevnar 13 is not," Petitioner's GI illness is "by far the likelier trigger." Id. at 17.

Lastly, with regard to timing, Dr. Whitton opined "coincidental temporal associations are inevitable" and "quite common." Resp. Ex. C at 16. He acknowledged Petitioner first experienced symptoms later diagnosed as GBS on approximately August 25, 2016, 15 days post-vaccination. Id. at 1; Resp. Ex. E at 1. He did not provide an opinion as to whether this timing is medically appropriate.

III. DISCUSSION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, Petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

Petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 57 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell ex rel. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 57 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded to medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health &

Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege he suffered a Table Injury, he must prove a vaccine he received actually caused his injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The special master must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and

the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

IV. ANALYSIS

A. Causation

1. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 257 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen, 618 F.3d at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

Here, the experts do not dispute the theory of molecular mimicry generally, nor do they dispute it as a theory that can cause disease or GBS in certain instances. They agreed infectious illnesses can precede GBS and that a GI illness can cause GBS. However, the experts dispute whether the Prevnar 13 vaccine can cause GBS via molecular mimicry.

Due to the facts and circumstances of this case, specifically the fact that Petitioner reported a diarrheal illness prior to the onset of his GBS, the undersigned's determination as to causation turns on an analysis of Althen prong two. Assuming that Petitioner has proven a sound

and reliable causal mechanism under Althen prong one,[68] the undersigned finds Petitioner has not provided preponderant evidence of a logical sequence of cause and effect because the evidence establishes he had a diarrheal illness antecedent to onset of his illness. Thus, the undersigned turns her focus to Althen prong two. See Vaughan ex rel. A.H. v. Sec'y of Health & Hum. Servs., 107 Fed. Cl. 212, 221-22 (2012) (finding the special master's failure to rule on Althen prong one not fatal to his decision because Althen prong two was fatal to the petitioner's case); Hibbard v. Sec'y of Health & Hum. Servs., 698 F.3d 1355, 1364 (Fed. Cir. 2012) ("discern[ing] no error in the manner in which the special master chose to address the Althen [prongs]" when he focused on Althen prong two after "assuming the medical viability of [the] theory of causation").

2. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

A petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and

[68] The undersigned is quite familiar with Dr. Steinman's theory presented in this case as it has previously been accepted as sound and reliable in at least 10 other Prevnar 13 cases, decided by different special masters, including the undersigned. See, e.g., Simeneta v. Sec'y of Health & Hum. Servs., No. 18-859V, 2024 WL 4881411, at *30-33 (Fed. Cl. Spec. Mstr. Oct. 31, 2024); Bartoszek v. Sec'y of Health & Hum. Servs., No. 17-1254V, 2024 WL 4263604, at *17-22 (Fed. Cl. Spec. Mstr. Aug. 27, 2024); Byrd v. Sec'y of Health & Hum. Servs., No. 20-1476V, 2024 WL 2003061, at *21-26 (Fed. Cl. Spec. Mstr. July 8, 2024); Cooper v. Sec'y of Health & Hum. Servs., No. 18-1885V, 2024 WL 1522331, at *14-18 (Fed. Cl. Spec. Mstr. Mar. 12, 2024); Anderson ex rel. Meyer v. Sec'y of Health & Hum. Servs., No. 18-484V, 2024 WL 557052, at *30-32 (Fed. Cl. Spec. Mstr. Jan. 17, 2024); Parker v. Sec'y of Health & Hum. Servs., No. 20-411V, 2023 WL 9261248, at *20-22 (Fed. Cl. Spec. Mstr. Dec. 20, 2023); Sprenger v. Sec'y of Health & Hum. Servs., No. 18-279V, 2023 WL 8543435, at *18-20 (Fed. Cl. Spec. Mstr. Nov. 14, 2023); Gross v. Sec'y of Health & Hum. Servs., No. 17-1075V, 2022 WL 9669651 (Fed. Cl. Spec. Mstr. Sept. 22, 2022); Maloney v. Sec'y of Health & Hum. Servs., No. 19-1713V, 2022 WL 1074087 (Fed. Cl. Spec. Mstr. Mar. 17, 2022); Pierson v. Sec'y of Health & Hum. Servs., No. 17-1136V, 2022 WL 322836, at *27-31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); Koller v. Sec'y of Health & Hum. Servs., No. 16-439V, 2021 WL 5027947, at *18 (Fed. Cl. Spec. Mstr. Oct. 8, 2021). The undersigned recognizes that there is not uniformity between the special masters in decisions addressing the Prevnar 13 vaccine and GBS. The undersigned further recognizes that prior decisions are not binding on the undersigned and can be considered by the undersigned in forming her opinions. See Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999); Boatmon, 941 F.3d at 1358.

effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In determining whether Petitioner has established a prima facia case, the undersigned finds it relevant to consider "evidence of other possible sources of injury" in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Stone, 676 F.3d at 1379. "In asserting an off-Table injury, [Petitioner] need[s] to show, by preponderant evidence, that his [Prevnar 13] vaccination was a substantial factor in causing his GBS." Winkler v. Sec'y of Health & Hum. Servs., 88 F.4th 958, 962 (Fed. Cir. 2023). Petitioner "[does] not need to show that he did not suffer from a [GI] infection, or that said [GI] infection did not contribute to his GBS. Nor [does] he have to show that the [Prevnar 13] vaccination was the only cause of his GBS." Id.; see also Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (finding a petitioner does not bear the burden of eliminating alternative independent potential causes). Thus, the undersigned considers evidence relating to whether Petitioner suffered from a diarrheal illness, as well as the likelihood that said illness triggered Petitioner's GBS, as "[s]uch contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law." Winkler, 88 F.4th at 963; see also Flores, 115 Fed. Cl. at 162-63 ("[T]he special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case.").

i. There Is Preponderant Evidence That Petitioner Had a Diarrheal Illness Prior to GBS Onset

First, the undersigned summarizes the relevant facts. Petitioner received a Prevnar 13 vaccination on August 10, 2016. He was noted to be well on the day of vaccination. On August 29, 2016, 19 days after vaccination, Petitioner reported abnormal sensations of his feet that started the day before. The following day, August 30, Petitioner reported diffuse myalgias and he appeared tired. On September 2, 2016, Petitioner returned to Fort Harrison "reporting tingling/numbness of feet and []9/10 pain of rest of body [for] [one] week." Pet. Ex. 3 at 145.

Petitioner presented to the emergency department on September 3, where he was seen by emergency physician Dr. Cohen on admission. Dr. Cohen documented, "Patient reports diarrhea several weeks ago." Pet. Ex. 6 at 5.

After he was admitted, Petitioner saw hospitalist Dr. Katz. Dr. Katz documented Petitioner's history, noting Petitioner reported "that over [two] weeks ago he had a brief diarrheal illness that has completely resolved." Pet. Ex. 6 at 20.

While hospitalized, Petitioner was seen in consultation by neurologist Dr. Knappenberger. Dr. Knappenberger wrote Petitioner "developed a mild GI illness with diarrhea for a couple of days, no fevers, no abdominal pain" one month ago. Pet. Ex. 6 at 24. Dr. Knappenberger also noted that Petitioner received a Prevnar 13 vaccination "about [three] weeks ago." Id.

Petitioner does not deny having diarrhea prior to his vaccination. See Pet. Ex. 160 at 2-3.

Therefore, the undersigned finds there is preponderant evidence that Petitioner experienced a diarrheal illness approximately one month prior to his hospitalization.

ii. Three of Petitioner's Treating Physicians Associated His Diarrheal Illness with His GBS

In evaluating whether this prong is satisfied, the opinions and views of Petitioner's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)).

The undersigned also finds that during his acute hospitalization, three of Petitioner's physicians, Dr. Cohen, Dr. Katz, and Dr. Knappenberger, documented Petitioner's history of having a diarrheal illness in association with his GBS. Dr. Cohen documented that Petitioner reported diarrhea several weeks before. Dr. Katz specifically opined that "[g]iven recent history of a diarrheal illness followed by development of the [reported] symptoms[,] [I] feel this is consistent with [GBS]." Pet. Ex. 6 at 23. Dr. Knappenberger noted both the diarrheal illness and vaccination as antecedent events to Petitioner's GBS.

Not all of Petitioner's treating physicians associated or attributed Petitioner's GBS to his diarrheal illness; some referenced the Prevnar 13 vaccination as causal. These records, however, occurred later in time, following Petitioner's discharge from the hospital. For example, after discharge from the hospital, Petitioner returned to see his PCP, Dr. Swoboda, who "strongly suspect[ed] GB[S] due to [Prevnar 13]." Pet. Ex. 3 at 55. He also listed Prevnar as an allergy/adverse reaction. Dr. Swoboda did not document Petitioner's diarrheal illness. Specifically, Dr. Swoboda did not document Petitioner's reported history of an antecedent diarrheal illness either before or after Petitioner's hospitalization and/or diagnosis of GBS. Therefore, based on his records, it is not clear whether Dr. Swoboda was aware of Petitioner's GI illness prior to the onset of his GBS.

Although treating physician statements are typically "favored," no treating physician's views bind the special master, per se; rather, their views are carefully considered and evaluated. § 13(b)(1); Capizzano, 440 F.3d at 1326; Snyder, 88 Fed. Cl. at 746 n.67. "As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases." Welch v. Sec'y of Health & Hum. Servs., No. 18-494V, 2019 WL 3494360, at *8 (Fed. Cl. Spec. Mstr. July 2, 2019). An opinion by a treating physician that is not supported by a factual basis or other evidence is conclusory in nature. See Robertson v. Sec'y of Health & Hum. Servs., No. 18-554V, 2022 WL 17484980, at *17 (Fed. Cl. Spec. Mstr. Dec. 7, 2022); Cedillo v. Sec'y of Health & Hum. Servs., 617 F.3d 1328, 1347 (Fed. Cir. 2010). Because it is not clear that Dr. Swoboda was aware of the antecedent diarrheal illness, the undersigned questions his opinions suggesting an association between GBS with vaccination.

Also following his hospitalization, Petitioner saw Dr. Knappenberger in September and October 2016. At these visits following hospitalization, Dr. Knappenberger's assessment was "[GBS] following vaccination." Pet. Ex. 2 at 27, 49. But the next two visits, on December 1, 2016 and February 2, 2017, do not mention vaccination. See id. at 62, 104.

The change in documentation by Dr. Knappenberger after hospitalization, to omit the references to a diarrheal illness, may be consistent with Petitioner's explanation.[69] However, Dr. Knappenberger does not offer any explanation in his record, and therefore, it would be speculative to draw conclusions based on Petitioner's description that Dr. Knappenberger had a "change in countenance."

In conclusion, the undersigned finds that some of Petitioner's treating physicians associated and/or attributed his GBS to his prior diarrheal illness.

iii. Petitioner's Experts Agree Petitioner's Diarrheal Illness Could Have Been Causative or a Substantial Contributing Factor

Both Dr. Nadareishvili and Dr. Steinman opined Petitioner's diarrhea could have been causative and/or a substantial contributing factor. Dr. Nadareishvili opined that he "[could not] entirely rule out a diarrheal illness as a potential cause." Pet. Ex. 99 at 17; see also Pet. Ex. 101 at 6 ("I cannot entirely rule out the potential contributory role of a diarrheal illness as a concomitant significant factor."). He further opined that the diarrheal illness "could potentially have been causative." Pet. Ex. 99 at 17.

Similarly, Dr. Steinman concluded the diarrheal illness was "evidence of [a] substantial factor that could contribute to the triggering of GBS." Pet. Ex. 142 at 29. He opined both the diarrheal illness and Prevnar 13 vaccine were "substantial factors" in causing Petitioner's GBS. Pet. Ex. 112 at 5, 30.

But both Dr. Nadareishvili and Dr. Steinman also opined there was insufficient evidence to support a finding that Petitioner's diarrheal illness was causative. The undersigned takes issue with their reasoning.

First, Dr. Nadareishvili opined the temporal association between vaccination and GBS was "very strongly supportive of the vaccination as a cause." Pet. Ex. 99 at 17. However, he did not acknowledge the same could be said about the diarrheal illness. The medical literature consistently identifies a diarrheal illness as an antecedent event in GBS. Thus, for Dr. Nadareishvili to favor vaccination over a diarrheal illness without a factual or other evidentiary basis renders his opinion less credible.

Second, Dr. Nadareishvili and Dr. Steinman cite to the fact that there was no testing to confirm a specific diarrheal illness as support that the Prevnar 13 vaccine was the more likely cause of Petitioner's GBS. Although accurate, this argument does not explain how the vaccination was the likely cause of Petitioner's GBS. Furthermore, "given th[e] lack of dispute

[69] See Pet. Ex. 160 at ¶ 12.

regarding [diarrheal illness] as a possible source of injury, evaluating the strength of [Petitioner's] prima facie case [does] not require an explicit finding that [Petitioner] actually suffered from a [specific] infection." Winkler, 88 F.4th at 963.

Third, Dr. Nadareishvili cited to treating physician statements, arguing the treating physicians opined the Prevnar 13 vaccine was the likely trigger for Petitioner's GBS. However, as described above, several of Petitioner's treating physicians documented as association between GBS and Petitioner's diarrheal illness.

iv. Respondent's Experts Opine That GBS Can Be Preceded by a GI or Diarrheal Illness

Respondent's experts, Dr. Chaudhry and Dr. Whitton, explain that approximately two-thirds of GBS cases are preceded by a GI or respiratory illness and cited supportive literature. See, e.g., Resp. Ex. A, Tab 1 at 1-2 ("Two-thirds of adult patients report preceding symptoms of a respiratory or [GI] tract infection within [four] weeks of onset of weakness."); Resp. Ex. C, Tab 11 at 3 tbl.1 (identifying an infectious agent in 56% of GBS patients); Resp. Ex. C, Tab 12 at 3 fig., 4.

Both Dr. Chaudhry and Dr. Whitton opine Petitioner had a diarrheal illness in the two to four weeks prior to neurologic symptoms onset. Dr. Chaudhry opined this course was consistent with a "rather typical antecedent history" seen in GBS patients. Resp. Ex. A at 7. And Dr. Whitton opined that because "GI infections are known to trigger GBS[] [and] Prevnar 13 is not," Petitioner's GI illness is "by far the likelier trigger." Resp. Ex. C at 17.

v. Conclusion

Overall, the undersigned is not persuaded by Petitioner's arguments, given Petitioner's clinical course, treating physician statements, and the experts' opinions supported by medical literature. The undersigned acknowledges that Petitioner is not required to eliminate other potential causes in order to be entitled to compensation. See Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (finding petitioner does not bear the burden of eliminating alternative independent potential causes). However, she finds it reasonable to consider "evidence of other possible sources of injury"—here, Petitioner's diarrheal illness—in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Stone, 676 F.3d at 1379.

In this case, "the presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination." Pafford, 451 F.3d at 1358-59; see also Walther, 485 F.3d at 1151 n.4 ("Where multiple causes act in concert to cause the injury, proof that a particular vaccine was a substantial cause may require the petitioner to establish that the other causes did not overwhelm the causative effect of the vaccine."). As such, the undersigned finds Petitioner failed to prove that the Prevnar 13 vaccine was the "but for" cause of Petitioner's GBS.

A recent Federal Circuit Decision in Winkler affirmed the undersigned's reliance on "evidence of other possible sources of injury"—in Winkler, a GI illness—in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Winkler, 88 F.4th at 962-63 (quoting Stone, 676 F.3d at 1379). Like Petitioner here, the petitioner in Winkler had a diarrheal illness around the time of vaccination and prior to developing GBS. Id. at 961. Here, and in Winkler, there is no dispute among the experts that the Petitioner had a diarrheal illness and that diarrheal illness was a possible source of injury. Id. at 963. The undersigned dismissed the petition in Winkler because she found petitioner did not provide preponderant evidence that the vaccine was a substantial factor in causing his GBS. Id. at 961. The Federal Circuit affirmed, noting "contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law." Id. at 963.

For the reasons described above, the undersigned finds Petitioner has not proven by preponderant evidence a logical sequence of cause and effect establishing that the Prevnar 13 vaccination caused Petitioner's GBS. Thus, Petitioner has not satisfied the second Althen prong.

3. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That phrase has been defined as a "medically acceptable temporal relationship." Id. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1579, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542.

The experts agree that Petitioner received his Prevnar 13 vaccination on August 10, 2016 and developed neurologic symptoms on approximately August 25, 2016, 15 days post-vaccination.

Petitioner's experts opine this timing is medically appropriate and consistent with the literature. Haber et al., for example, reported 11 cases of GBS following a Prevnar 13 vaccine, with a median onset interval of nine days and a range of two to 42 days. Although a study on the flu vaccine and not Prevnar 13, Schonberger et al. reported peak GBS onset two to three weeks post-vaccination. Respondent's experts did not advance any arguments to the contrary or otherwise dispute that there was a temporal association between vaccination and GBS onset consistent with the theory of molecular mimicry.[70]

This time frame from vaccination to the initial manifestation of symptoms is appropriate given the theory of molecular mimicry. This temporal association is also consistent with the

[70] Respondent's experts do argue Petitioner's date of symptom onset is consistent with a diarrheal illness as a cause of GBS. Petitioner's experts do not appear to dispute this.

onset period of three to 42 days as set forth in the Vaccine Injury Table for GBS following flu vaccination. 42 C.F.R. § 100.3(a)(XIV)(D).

Further, this time frame has been acknowledged as appropriate in other Vaccine Program cases in which molecular mimicry has been proffered as the causal mechanism. See, e.g., Simeneta, 2024 WL 4881411, at *34-35 (finding a GBS onset of 18 days after Prevnar 13 vaccination to be appropriate); Bartoszek, 2024 WL 4263604, at *24-25 (finding a GBS onset of 22 days, or approximately three weeks, post-Prevnar 13 vaccination to be medically acceptable); Sprenger, 2023 WL 8543435, at *22 (finding a GBS onset of approximately two weeks after Prevnar 13 vaccination to be appropriate); Gross, 2022 WL 9669651, at *38-39 (finding a GBS onset of 13 days after Prevnar 13 vaccination to be appropriate); Koller, 2021 WL 5027947, at *23 (finding a GBS onset of 12 days after Prevnar 13 vaccination to be "within the medically accepted timeframe consistent with [P]etitioner's theory of molecular mimicry [and] that has been accepted in other Vaccine Program cases"); see also Barone v. Sec'y of Health & Hum. Servs., No. 11-707V, 2014 WL 6834557, at *13 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) ("[S]pecial masters have never gone beyond a two-month (meaning eight week) interval in holding that a vaccination caused a demyelinating illness.").

However, a temporal association, without more, is insufficient. Moberly, 592 F.3d at 1323; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1148 (Fed. Cir. 1992) ("[A] proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury."). Thus, even though Petitioner has provided preponderant evidence satisfying Althen prong three, Petitioner is not entitled to compensation.

V. CONCLUSION

The undersigned extends her sympathy for the suffering Petitioner has experienced due to his illness. However, the undersigned's Decision cannot be based on her sympathy, but must be based on the evidence and the law.

For the reasons discussed above, the undersigned finds that Petitioner has failed to provide preponderant evidence of causation, and therefore, the petition must be dismissed.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master